104. Postmen

Table 104 gives details of the studies which investigated a possible association between lung cancer risk and employment as a postman. Nine standardized mortality ratios, lying in the range 96-123, were calculated with all but one being raised. Two proportional mortality ratios, of 99 and 102, were given along with two proportional registration ratios of 111 and 118.

Although the data in the table appear to show a higher risk of lung cancer among postmen, the increase in risk does not appear to be very large. No data was available on possible exposures postmen might be subjected to, and there was no attempt by any of the studies to explain the observed increase in lung cancer risk. It should also be remembered that the data came from very few studies. Therefore, due to a lack of information it is not really possible to draw any firm conclusions about the risk of lung cancer among postmen.

Table 104: Estimates of standardized mortality ratio for employment as a postman

Study	Population	Standardized mortality ratio
OPCS (1978)	English/Welsh postmen/mail sorters, aged 15-64	99*
	Aged 65-74	102*
	Incidence 1966-7	118($p<0.01$)[1]
	1968-9	111[1]
Logan (1982)	English/Welsh postmen, 1931	110
	1951	113
	1961	117
	1971	96
	Married women[2], 1951	121
	1961	123
	1971	111
OPCS (1986)	UK male postmen/mail sorters/messengers	104
	Married women[2]	116

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Footnote to Table 104

* Proportional mortality ratio

1 Proportional registration ratio

2 According to husband's occupation

References

1. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

2. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

3. Office of Population Censuses and Surveys (1986) Occupational mortality: The Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83. HMSO, London.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

105. Printers

Only two studies were found which attempted to relate the risk of lung cancer to employment in the printing industry, and details of these are given in Table 105. Four standardized mortality ratios between 91 and 141 were presented, of which three were raised.

Neither study attempted to explain their findings, and indeed one of them stated that their survey did not "provide any evidence about the cause of the overall small excess of deaths from lung cancer, which might or might not be occupational" [1]. With so few studies reporting, it is difficult to draw any firm conclusions about the carcinogenicity of exposures associated with the printing industry, but it is likely that if a risk does exist it is not very large.

Table 105: Estimates of standardized mortality ratio for workers in the printing industry

Study	Population	Standardized mortality ratio
Moss et al (1972)	UK male printing trade workers - London	132($p<0.01$)
	Manchester	141($p<0.01$)
OPCS (1986)	UK male printing workers/screen/block printers	106
	Married women[1]	91

1 According to husband's occupation

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

References

1. Moss E, Scott TS and Atherley GRC (1972) Mortality of newspaper workers from lung cancer and bronchitis 1952-66. Br J Ind Med, 29, 1-14.

2. Office of Population Censuses and Surveys (1986) Occupational mortality: The Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83. HMSO, London.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

106. Publicans

Table 106 gives details of the two studies which gave information relevant to an investigation of the possible association between lung cancer and employment as a publican. Seven standardized mortality ratios of between 104 and 197 were presented. Two proportional mortality ratios of 101 and 105 were given, along with two proportional registration ratios of 118 and 122.

No data was available on possible exposures of publicans, and there was no attempt to explain the observed increase in lung cancer risk. Although the evidence suggests an increased risk of lung cancer among publicans, with so little information available it is difficult to properly evaluate the potential carcinogenicity of such employment.

Table 106: Estimates of standardized mortality ratio for publicans

Study	Population	Standardized mortality ratio
OPCS (1978)	E/W male publicans/innkeepers, aged 15-64	105*
	Aged 65-74	101*
	Incidence 1966-7	118($p<0.05$)[1]
	1968-9	122($p<0.01$)[1]
Logan (1982)	E/W men, 1931	146
	1951	144
	1961	142
	1971	153
	Married women[2], 1951	104
	1961	197
	1971	156

E/W = English/Welsh

* Proportional mortality ratio

1 Proportional registration ratio

2 According to husband's occupation

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

References

1. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

2. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

107. Radiotherapy

Table 107 gives details of the two studies found which attempted to relate lung cancer risk to exposure to radiotherapy. Two relative risks, of 1.2 and 3.7, were estimated, along with a standardized mortality ratio of 142.

Only the study by Smith and Doll gave information on the doses of radiation received by respondents. Thus, in the study by Kabat inaccuracies could have been introduced due to misclassification of exposure. Furthermore, in this study there was a high correlation between a history of a reproductive primary and a history of radiotherapy, and it was not possible to estimate the effect of one exposure independent of the other.

In any case, with only two studies reporting it is difficult to evaluate the potential carcinogenicity of radiotherapy to humans.

Table 107: Estimates of relative risk/standardized mortality ratio for exposure to radiotherapy

Study	Population	Relative risk (95% limits)
Doll (1981)/Smith and Doll (1982)	UK ankylosing spondylitis patients	142(p<0.001)*
Kabat (1993)	US male radiotherapy patients	1.2(0.2-6.4)
	Females	3.7(1.2-10.9)

* Standardized mortality ratio

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

References

1. Doll R (1981) Radiation hazards: 25 years of collaborative research. Brit J Radiol, 54, 179-186.

2. Kabat GC (1993) Previous cancer and radiotherapy as risk factors for lung cancer in lifetime nonsmokers. Cancer Causes and Control, 4, 489-495.

3. Smith PG and Doll R (1982) Mortality among patients with ankylosing spondylitis after a single treatment course with x rays. Brit Med J, 284, 449-60.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

108. Radon

Lack of time meant that a full evaluation of the evidence relating lung cancer risk to radon exposure could not be made. However, both IARC and the National Research Council reviewed this area in 1988 [1,2]. Although radon is ubiquitous, concentrations to which humans may be exposed in enclosed areas, such as underground mines and in buildings, are elevated compared to levels in outdoor air. Evidence for an increased risk of lung cancer following exposure to radon comes from a number of cohort and case-control studies of underground miners, including particularly uranium miners, but also iron-ore and other metal miners, and one group of fluorspar miners. Evidence for dose-response relationships has been obtained from several of these studies. Additionally, a higher risk of lung cancer among individuals living in houses known or presumed to have higher levels of radon than among those with lower presumed exposure has been suggested by several case-control studies, although these have been based on small numbers of subjects. There is also some evidence that the interaction of radon and its decay products with cigarette smoking with regard to lung cancer may follow a multiplicative or submultiplicative model [1,2].

Overall, IARC felt that there was "sufficient" evidence of the carcinogenicity of radon and its decay products to humans [1].

References

1. International Agency for Research on Cancer (1988) Monographs on the evaluation of carcinogenic risks to humans. Volume 43: Man-made mineral fibres and radon, 173-259. IARC, Lyon.

2. National Research Council (1988) Health risks of radon and other internally deposited alpha-emitters: BEIR IV. National Academy Press, Washington DC.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

109. Rail workers

Only three studies were found which investigated a possible association between the risk of lung cancer and rail workers, and details of these are given in Table 109. The nine standardized mortality ratios presented ranged from 83-115, with five of them being raised.

It has been suggested that rail workers may be exposed to a number of compounds which may be carcinogenic, particularly asbestos [1,2]. The study by Howe attempted to relate the risk of lung cancer in rail workers to exposure to diesel fumes and coal dust, but estimates of the level of exposure were used in place of objective measurements. Apart from this, no information was available on the subjects' exposures, so it is not possible to determine the substances to which they may have been exposed, or the possible role of any one substance in the development of lung cancer.

Thus, it is very difficult to evaluate the evidence for the carcinogenicity of exposures of rail workers but from the data presented in the table a clear pattern of increased risk does not really emerge and therefore any risk, if it does exist, is probably not very large.

Table 109: Estimates of standardized mortality ratio for rail workers

Study	Population	Standardized mortality ratio
Logan (1982)	English/Welsh male railway porters - 1931	100
	1951	108
	1961	106
	1971	115
	Married women[1] - 1961	95
	1971	115
Howe et al (1983)	Canadian male rail workers	106
OPCS (1986)	UK male rail transport operating staff	87
	Married women[1]	83

1 According to husband's occupation

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

References

1. Garshick E, Schenker MB, Munoz A et al (1987) A case-control study of lung cancer and diesel exhaust exposure in railroad workers. Am Rev Respir Dis, 135, 1242-1248.

2. Howe GR, Fraser D, Lindsay J, Presnal B and Yu SZ (1983) Cancer mortality (1965-1977) in relation to diesel fume and coal exposure in a cohort of retired railway workers. JNCI, 70, 1015-1019.

3. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

4. Office of Population Censuses and Surveys (1986) Occupational mortality: The Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83. HMSO, London.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

110. Rationality/antiemotionality

Only two studies were found which attempted to relate lung cancer risk to measures of rationality and antiemotionality, and details of these are given in Table 110. One study reported that all 38 lung cancer cases occurred in those scoring highly for rationality and antiemotionality, compared to 11.62 expected, giving a standardized mortality ratio of 3.27, while the other reported that lung cancer patients showed higher prevalences of traits associated with rationality and antiemotionality than controls.

The study by Grossarth-Maticek has attracted much criticism since it was published, and the results are generally considered to be seriously flawed due to drawbacks in the study design and the method of recruiting respondents. Thus, with only one other study reporting it is not possible to evaluate the relationship between lung cancer risk and rationality and antiemotionality.

Table 110: Estimates of standardized mortality ratio for rationality/ antiemotionality

Study	Population	Standardized mortality ratio (95% limits)
Blohmke et al (1984)	German/Austrian residents	Lung cancer patients showed lack of nervousness, high social conformity and strong external control compared to controls
Grossarth-Maticek et al (1985)	Yugoslavian residents	3.27($p<0.001$)

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

References

1. Blohmke M, von Engelhardt B and Stelzer O (1984) Psychosocial factors and smoking as risk factors in lung carcinoma. J Psychosomatic Res, 28, 221-229.

2. Grossarth-Maticek R, Bastiaans J and Kanazir DT (1985) Psychosocial factors as strong predictors of mortality from cancer, ischaemic heart disease and stroke: The Yugoslav prospective study. J Psychosomatic Res, 29, 167-176.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

111. Respiratory symptoms

Details of studies which attempted to relate the risk of lung cancer to the presence of respiratory symptoms are shown in Table 111. Relative risks ranging from 0.8-3.23 were estimated, with thirteen out of fourteen being above 1.00.

Due to the reported associations between cigarette smoking, respiratory symptoms and lung cancer, all of the studies collected information on the respondents' smoking habits. However, as noted by Wynder and Fairchild, due to the strong correlation in this study between cigarette smoking and lung cancer and cigarette smoking and persistent cough, it was difficult to properly evaluate the correlation of cough to lung cancer, independent of cigarette smoking.

Therefore, despite the evidence presented in the table suggesting a possible association between the presence of respiratory symptoms and lung cancer risk, such a relationship cannot be regarded as established.

Table 111: Estimates of relative risk for respiratory symptoms

Study	Population	Symptoms	Relative risk (95% limits)
Denoix et al (1958)	French men	BRON	2.00(0.95-4.22)[1]
Wynder and Fairchild (1965)	US men	BRON	3.23(1.61-6.48)[1]
		COU	2.76[1](p<0.001)
		FLU	1.24(0.71-2.16)[1]
		PND	1.87(1.09-3.20)[1]
		SINU	1.85(1.05-3.26)[1]
Tenkanen et al (1987)	Finnish men	PHL	2.0(p<0.001)
		SOB	2.2(p<0.001)
		WHE	2.0(p<0.001)
Vestbo et al (1991)	Danish men	BRON	0.8(0.3-2.7)[2]
		COU	2.5(1.3-5.0)[2]
		PHL	1.5(0.7-3.2)[2]
		PHLC	1.2(0.5-3.0)[2]
		SOB	2.2(1.0-4.9)[2]

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Footnote to Table 111

BRON = bronchitis; COU = cough; FLU = influenza; PHL = phlegm; PHLC = chronic phlegm; PND = postnasal drip; SINU = sinusitis; SOB = shortness of breath; WHE = wheezing

1 Estimated from data given

2 Tumours of respiratory system

References

1. Denoix PF, Schwartz D and Anguera G (1958) French investigation into the etiology of broncho-pulmonary cancer. Detailed analysis. Bull Assoc Franc pour l'etude du cancer, 49: 1-37.

2. Tenkanen L, Hakulinen T and Teppo L (1987) The joint effect of smoking and respiratory symptoms on risk of lung cancer. Int J Epidemiol, 16, 509-515.

3. Vestbo J, Knudsen KM and Rasmussen FV (1991) Are respiratory symptoms and chronic airflow limitation really associated with an increased risk of respiratory cancer? Int J Epidemiol, 20, 375-378.

4. Wynder EL and Fairchild EP (1965) The role of a history of persistent cough in the epidemiology of lung cancer. Am Rev Resp Dis, 94, 709-720.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

112. The rubber industry

Details of the studies which investigated lung cancer risk amongst workers in the rubber industry are given in Table 112. Eighteen standardized mortality ratios, ranging between 47 and 434, were given, with 12 being raised. Five relative risks, of 1.4-2.3, were estimated, and one study also gave an incidence ratio of 126.

Workers in the rubber industry are likely to be exposed to a combination of chemicals, in the form of dusts, vapours, condensed volatiles, solvents and gases, which will vary depending on the stage of the manufacturing process. Many of the materials which occur in rubber factories have been shown experimentally to be mutagenic or carcinogenic, and these include mineral oils, carbon black (extracts), curing fumes, some monomers, solvents, nitroso compounds and aromatic amines, thiurams and dithiocarbamate compounds, ethylenethiourea, di(2-ethylhexyl) phthalate, di(2-ethylhexyl) adipate and hydrogen peroxide [2]. However, due to a lack of historical industrial hygiene data none of the studies could accurately ascertain exposure, although some (Baxter and Werner, Delzell, Fox, McMichael 1976, Monson and Fine, Monson and Nakano 1976b, Parkes, Zhang) did attempt to classify workers into particular job categories as a substitute for exposure categories. Thus, attempting to evaluate the potential carcinogenicity of any one compound will be extremely difficult.

In their assessment of the rubber industry in 1982, IARC stated that "the combination of chemical exposures that occurs in the rubber industry is probably more relevant to the cancer pattern observed than are single compounds or groups of compounds. The variety of exposures increases the likelihood that there are interactive effects between two or more such agents" [2]. It was felt that there was "sufficient" evidence for the carcinogenicity in humans of exposures associated with the rubber industry [2,3].

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Table 112: Estimates of relative risk/standardized mortality ratio for workers in the rubber industry

Study	Population	Relative risk (95% limits)
Fox et al (1974)	British rubber workers	118*
McMichael et al (1974)[1]	US rubber workers	83*
Andjelkovich et al (1976)[1]	US rubber workers	83*
Fox and Collier (1976)[1]	British rubber workers	127*
McMichael et al (1976a,b)[1]	US Receivers/shippers	1.9[2]
	Compounders/mixers	1.4[2]
	Mill-mixers	2.1[2]
	Extruders	1.4[2]
	Reclaimers	2.3[2]
Monson and Nakano (1976a)[1]	US rubber workers	92*
Monson and Nakano (1976b)[1]	US female non-tyre rubber workers	333*
Andjelkovich et al (1977)[1]	US workers exposed to synthetic latex	434*
Andjelkovich et al (1978)[1]	US female rubber workers	191*[3]
Monson and Fine (1978)[1]	US tyre curers	220*[3]
	Tyre moulders	200*[3]
	Fuel cell/deicer manufacturers	158*[3]
Baxter and Werner (1980)[1]	British rubber workers	115*[3]
Bovet and Lob (1980)[1]	Swiss rubber workers	47*
Delzell and Monson (1981)[1]	US rubber workers	84*
Kilpikari et al (1981)[1]	Finnish rubber workers	150*[2]
Delzell et al (1982)[1]	US tyre workers - mortality	99*
	Incidence	126
Parkes et al (1982)[1]	British rubber workers	122*
Zhang et al (1989)	Chinese rubber workers	133*

* Standardized mortality ratio

1 From IARC (1982)

2 Tumours of respiratory system

3 Estimated from data given

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

References

1. Fox AJ, Lindars DC and Owen R (1974) A survey of occupational cancer in the rubber and cablemaking industries: Results of five-year analysis, 1967-71. Br J Ind Med, 31, 140-151.

2. International Agency for Research on Cancer (1982) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 28: The rubber industry. IARC, Lyon.

3. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogencity: An updating of IARC monographs volumes 1 to 42, 332-334. IARC, Lyon.

4. Zhang Z-F, Yu S-Z, Li W-X and Choi BCK (1989) Smoking, occupational exposure to rubber, and lung cancer. Br J Ind Med, 46, 12-15.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

113. Schizophrenia

Table 113 gives details of the three studies which investigated the risk of lung cancer in relation to schizophrenia. A relative risk of 0.33 was given for lung cancer incidence, while the other two studies reported that lung cancer mortality among schizophrenics was either equal to or lower than that of other populations.

One author (Mortensen) felt that the lower risk of lung cancer observed could be due to a reduced exposure to carcinogens such as cigarette smoke among schizophrenic patients. However, the other two commented on the fact that the patients in their studies showed a high prevalence of smoking, of up to 90%. In the light of this, the reduced incidence of lung cancer seems even more surprising.

While the evidence presented in the table appears to suggest a reduced risk of lung cancer among schizophrenic patients, with only three studies reporting it is difficult to make a full evaluation.

Table 113: Estimates of relative risk for schizophrenic patients

Study	Location	Relative risk (95% limits)
Anon (1986)	USA	Schizophrenic patients had lower lung cancer mortality than controls
Gopalaswamy and Morgan (1986)	Not stated	Schizophrenics' lung cancer mortality no higher than general population
Mortensen (1989)	Denmark	0.33($p<0.001$)[1]

1 Incidence

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

References

1. Anon (1986) Br Med J, 293, 700.

2. Gopalaswamy AK and Morgan R (1986) Smoking in chronic schizophrenia. Br J Psych, 149, 523.

3. Mortensen PB (1989) The incidence of cancer in schizophrenic patients. J Epidemiol Community Health, 43, 43-47.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

114. Selenium

Table 114 gives details of the five studies which investigated the possible association between lung cancer risk and selenium levels.

A correlation study found evidence of lower lung cancer mortality rates in areas with higher selenium levels in forage crops, with mortality rates per 100,000 of 32.37, 33.25 and 40.06 for men in high, medium and low selenium areas respectively, and 5.45, 5.73 and 6.33 respectively for women.

Two of the studies measuring dietary selenium intake used high intake as a baseline. One of these studies estimated a relative risk of 6.00 for low selenium intake in one population considered, but failed to find an association, or to give detailed results for the other. The other study found lower selenium levels in lung cancer cases in three populations studied, but higher levels in cases in two populations. Again, detailed results were not presented.

A study of Dutch subjects, in which those with a high dietary intake of selenium were compared to those with a low intake, estimated a relative risk of 0.50.

Finally, a study of occupational selenium exposure found lower levels in the lung tissue of lung cancer cases than in that of two control groups.

The major drawback with the studies which investigated dietary selenium intake is that it is well known that selenium cannot be measured accurately by a dietary questionnaire alone, since the level in the food is dependent on the level in the soil in which it was grown. The study by van den Brandt measured selenium levels in the respondents' toenail clippings as well as administering a dietary questionnaire, thus providing a more objective measure of selenium. However, the studies by Comstock and Fontham did not appear to have taken any such measurements.

Another problem is that the development of cancer may affect appetite or the way in which food is metabolized, either of which could affect selenium levels measurable in the body. Therefore, the study by Fontham, using a case-control design in which information on selenium levels was collected after the subject had developed cancer may have introduced bias into the results. Similar problems may have occurred in the study by Gerhardsson.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Therefore, despite the findings presented in the table suggesting a possible protective effect of selenium against lung cancer, the relationship is far from proven. Indeed, after a recent review of the data IARC concluded that the evidence for such an effect was "not convincing" [3].

Table 114: Estimates of relative risk/mortality rate for selenium

Study	Population	Relative risk (95% limits)
Correlation study:		
Shamberger et al (1976)	US men, high selenium area	32.27[1]
	Medium selenium area	33.25[1]
	Low selenium area	40.06[1]
	Women, high selenium area	5.45[1]
	Medium selenium area	5.73[1]
	Low selenium area	6.33[1]
Low dietary selenium:		
Fontham (1990)	Not stated	No association reported
	Finnish residents	6.00
Comstock et al (1992)	Finnish males	Serum selenium levels lower in cases than controls ($p>0.05$)
	Finnish females	Serum selenium levels lower in cases than controls ($p>0.05$)
	US residents	Serum selenium levels lower in cases than controls ($p<0.05$)
	Hawaiian residents	Serum selenium levels higher in cases than controls ($p>0.05$)
	US residents	Serum selenium levels higher in cases than controls ($p>0.05$)

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Table 114 continued

High dietary selenium:		
Van den Brandt et al (1993a,1993b)	Dutch residents	0.50(0.30-0.81)
Occupational exposure:		
Gerhardsson et al (1985)	Swedish smelter workers	Selenium level in lung tissue 71ppb in cases compared to 110ppb and 136ppb in controls

1 Mortality rate per 100,000

References

1. Comstock GW, Bush TL and Helzlsouer K (1992) Serum retinol, beta-carotene, vitamin E, and selenium as related to subsequent cancer of specific sites. Am J Epidemiol, 135, 115-121.

2. Fontham ETH (1990) Protective dietary factors and lung cancer. Int J Epidemiol, 19 (Suppl 1), S32-S42.

3. Gerhardsson L, Brune D, Nordberg GF and Wester PO (1985) Protective effect of selenium on lung cancer in smelter workers. Br J Ind Med, 42, 617-626.

4. International Agency for Research on Cancer (1975) Monographs on the evaluation of carcinogenic risk of chemicals to man. Volume 9: Some aziridines, N-, S- and O-mustards and selenium, 245-260. IARC, Lyon.

5. Van den Brandt PA, Goldbohm RA, van't Veer P et al (1993a) Toenail selenium and the risk of lung, gastrointestinal, and breast cancer: A prospective study. Am J Epidemiol, 138, 626-627 (Abstract).

6. Van den Brandt PA, Goldbohm RA, van't Veer P et al (1993b) A prospective cohort study on selenium status and the risk of lung cancer. Cancer Res, 53, 4860-4865.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

115. Shellfish/crustaceae

Details of the only study found which attempted to relate the risk of lung cancer to consumption of shellfish and crustaceae are given in Table 115. It was reported that 36% of cases and 26% controls consumed cooked shellfish, while 16% of cases and 10% controls were consumers of crustaceae.

With only one study reporting it is not really possible to evaluate the potential carcinogenicity to humans of shellfish or crustaceae.

Table 115: Frequency of consumption of shellfish/crustaceae

Study	Population	Observations
Denoix et al (1958)	French men	36% bronchial cancer cases consumed cooked shellfish compared to 26% controls ($p<0.001$); 16% bronchial cancer cases consumed crustaceae compared to 10% controls ($p<0.01$)

References

1. Denoix PF, Schwartz D and Anguera G (1958) French investigation into the etiology of broncho-pulmonary cancer. Detailed analysis. Bull Assoc Franc pour l'etude du cancer, 49: 1-37.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

116. Silica

The numerous studies found which contained some data relevant to an investigation of the possible association between exposure to silica and the risk of lung cancer are detailed in Table 116. From the table, it can be seen that, of the 40 estimates of relative risk given ranging from 0.2-7.9, some 34 were above 1.00. The 26 standardized mortality ratios calculated lay in the range 84-590, with 22 of them being raised. Fourteen proportional mortality ratios were given, of between 75 and 348, of which 13 were raised. Additionally, one study gave an mortality odds ratio of 1.5.

From the table it can be seen that the study subjects came from many different occupational backgrounds. It is likely that the respondents were exposed to substances other than silica, which may themselves be potential carcinogens, in the course of their work and the nature of these exposures will vary from job to job. Data on the level of exposure to silica were only available from 13 of the studies (Davis, Gibson, Hessel and Sluis-Cremer, Hessel et al, Higgins, Kjuus, Koskela 1976, Lawler, McLaughlin, Meijers, Sherson and Iversen, Silverstein, Thomas and Stewart), and even fewer measurements of exposures to other substances were made. Therefore it is difficult to draw any firm conclusions about the role of silica in the development of lung cancer. Furthermore, all of the studies concentrated on the effects of crystalline silica, with none presenting results for amorphous silica.

Lastly, as IARC noted, there are some potential problems in the design of some of the studies. In particular there was some doubt as to the comparability of reference groups in several of the studies (Chiyotani, Costello and Graham, Davis, Pham, Vutuc, Westerholm 1986, Zambon), and the comprehensiveness with which some studies had taken into account the effects of possible confounding variables (Brown, Finkelstein 1982, Katsnelson and Mokronosova, Lawler, Muller, Neuberger, Selikoff, Thomas). Other problems which may have introduced inaccuracies into the studies, particularly those based on silicosis sufferers, included differences between countries in the definition of compensable silicosis, potential differences in disease detection methods, and unorthodox methods of calculating results [1,5].

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Therefore, it was felt that the evidence for the carcinogenicity to humans of crystalline silica was "limited", while that for amorphous silica was "inadequate" [5,6].

Table 116: Estimates of relative risk/standardized mortality ratio for exposure to silica

Study	Population	Relative risk (95% limits)
Redmond (1975)[1]	US masons	2.12($p<0.01$)
	Foundrymen	1.62
	Blacksmiths	2.42($p<0.05$)
Koskela et al (1976)[1]	Finnish foundry workers	151*
Milham (1976)[1]	US metal moulders	312[2]
	Mine operatives/labourers	179($p<0.05$)[2]
Gibson et al (1977)[1]	Canadian foundrymen	5.0($p<0.0005$)
Selikoff (1978)[3]	US tunnel workers	160*
Armstrong et al (1979)[3]	Australian gold miners	140($p<0.05$)*
Decoufle and Wood (1979)[1]	Iron foundry workers	200*
Egan et al (1979)[1]	US moulders - whites	147($p<0.01$)[2]
	Blacks	180[2]
Katsnelson and Mokronosova (1979)[1]	Gold miners - underground	7.9($p<0.001$)
	Surface miners	1.6
	Firebrick plant workers	2.0($p<0.05$)
Puntoni et al (1979)[1]	Italian sandblasters	3.8($p<0.05$)
Tola et al (1979)[1]	Finnish foundry workers	144($p<0.05$)[2]
Peterson and Milham (1980)[1]	US metal moulders	192($p<0.05$)[2,4]
	Mine operatives/labourers	138($p<0.05$)[2]
Westerholm (1980)[3]	Swedish miners/quarrymen/ tunnellers with silicosis diagnosed 1931-48	590($p<0.01$)*
	Diagnosed 1949-69	380($p<0.01$)*
	Iron/steel workers with silicosis diagnosed 1949-69	220($p<0.05$)*
Egan-Baum et al (1981)[1]	Iron foundry workers	2.4(1.01-5.3)

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Table 116 continued

Redmond et al (1981)[1]	US blacksmiths	1.89(p<0.05)
Costello (1982)[1]	Metal ore miners	127(p<0.01)*
Finkelstein et al (1982)[1]	Canadian miners	198(p<0.01)*
Fletcher (1982)[1]	English foundrymen	147(120-180)*
Milham (1982)[1]	Sandblasters	348(p<0.01)[2]
Thomas (1982)[1]	US pottery workers	180(p<0.01)[2]
Davis et al (1983)[3]	US granite workers	118*[5]
Higgins et al (1983)[3]	US taconite miners	84*
Lawler et al (1983)[3]	US haematite miners - surface	88*
	Underground	100*
Muller et al (1983)[3]	Canadian miners	145(p<0.001)*
Pham et al (1983)[3]	French iron miners	3.5(1.9-6.0)
Vutuc (1983)[3]	Austrian stone workers	2.0(p<0.01)
Gudbergsson et al (1984)[3]	Finnish silicotics	3.0(1.5-5.3)
Chiyotani (1984)[3]	Japanese silicotics	653
Puntoni et al (1985)[3]	Italian refractory plant workers with silicosis	167*
	Non-silicotics	208*
Rubino et al (1985)[3]	Italian silicotics	1.36(p<0.05)
Brown et al (1986)[3]	US gold miners	100*
Costello and Graham (1986)[3]	US granite workers	1.00
Finkelstein et al (1986)[3]	Canadian ceramic/pottery/ granite/quarry workers, silica brickmakers, sand-blasters with silicosis	302(p<0.01)*
Fletcher (1986)[3]	UK foundry workers	149(p<0.001)*
	Skilled moulders	125(p<0.05)[2]
	Semiskilled moulders	154(p<0.05)[2]
	Dressers	75[2]
	Labourers	106[2]
Forastiere et al (1986)[3]	Italian pottery workers	2.0(1.1-3.5)
Hessel and Sluis-Cremer (1986)[3]	South African miners	1.1
Kjuus et al (1986)[3]	Norwegian ferroalloy workers	1.00
Kurppa et al (1986)[3]	Finnish silicotics	312(230-414)*

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Table 116 continued

Lynge et al (1986)[6]	Finnish/Danish stone cutters	>1.00
	Swedish/Norwegian stone cutters	1.00
	Finnish excavation workers	>1.00
	Danish glass workers	>1.00
	Nordic ceramics workers	1.00
	Nordic metal miners	1.0-5.0
	Nordic foundry workers	>1.00
Neuberger et al (1986)[3]	Austrian silicotics	148(p<0.001)*
Schuler and Ruttner (1986)[3]	Swiss silicotics	2.41(p<0.05)
Sherson and Iversen (1986)[3]	Danish foundry workers	115*
Silverstein et al (1986)[3]	US iron foundry workers	148[2]
Steenland and Beaumont (1986)[3]	US members of Granite Cutters Union	1.19(0.97-1.5)
Westerholm et al (1986)[3]	Miners/quarrymen/tunnellers	4.1(p<0.05)
	Iron/steel workers	1.8
Zambon et al (1986)[3]	Italian silicotics	228(p<0.05)*
Koskela et al (1987)[6]	Finnish granite workers	129*[5]
Thomas and Stewart (1987)[6]	US pottery workers	137*
Mastrangelo et al (1988)[7]	Silicotics	1.9(p<0.05)
Forastiere et al (1989)[7]	Silicotics	1.5(p<0.05)[8]
Infante-Rivard et al (1989)	Canadian silicotics	3.47(3.11-3.90)
Amandus and Costello (1990)[7]	Miners with silicosis	2.0(p<0.05)
Hessel et al (1990)	South African gold miners	Cases and controls comparable for silica dust exposure
Meijers et al (1990)	Dutch ceramics workers	1.11(0.77-1.6)
Ng et al (1990)[7]	Silicotics	2.0(p<0.05)
Amandus et al (1991)	Workers in US dusty trades industry	2.3(1.5-3.4)
McLaughlin et al (1992)	Pottery workers	2.2(0.8-6.3)[5]
	Tungsten miners	0.5(0.3-1.0)
	Iron-copper miners	0.2(0.1-0.5)
	Tin miners	2.5(1.3-4.8)

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Footnote to Table 116

* Standardized mortality ratio

1 From Goldsmith (1982)

2 Proportional mortality ratio

3 From International Agency for Research on Cancer (1987a)

4 Tumours of respiratory system

5 Estimated from data given

6 From International Agency for Research on Cancer (1987b)

7 From Amandus (1991)

8 Mortality odds ratio

References

1. Amandus HE, Shy C, Wing S, Blair A and Heineman EF (1991) Silicosis and lung cancer in North Carolina dusty trades workers. Am J Ind Med, 20, 57-70.

2. Goldsmith DF, Guidotti TL and Johnston DR (1982) Does occupational exposure to silica cause lung cancer? Am J Ind Med, 3, 423-440.

3. Hessel PA, Sluis-Cremer GK and Hnizdo E (1990) Silica exposure, silicosis, and lung cancer: a necropsy study. Br J Ind Med, 47, 4-9.

4. Infante-Rivard C, Armstrong B, Petitclerc M, Cloutier L-G and Theriault G (1989) Lung cancer mortality and silicosis in Quebec, 1938-85. The Lancet, Dec 23/30, 1504-1507.

5. International Agency for Research on Cancer (1987a) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 42: Silica and some silicates, 39-144. IARC, Lyon.

6. International Agency for Research on Cancer (1987b) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC mongraphs volumes 1 to 42, 341-343. IARC, Lyon.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

7. McLaughlin JK, Chen J-Q, Dosemeci M et al (1992) A nested case-control study of lung cancer among silica exposed workers in China. Br J Ind Med, 49, 167-171.

8. Meijers JMM, Swaen GMH, Volovics A, Slangen JJM and Van Vliet K (1990) Silica exposure and lung cancer in ceramic workers: A case-control study. Int J Epidemiol, 19, 19-25.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

117. Smoked/salted/cured/pickled food

Only one study could be found which attempted to relate lung cancer risk to frequent consumption of foods which were smoked, salted, cured or pickled, and details of it are given in Table 117. Three relative risks, ranging from 0.92-1.60, were estimated for various food items.

It can be seen from the evidence presented in the table that there is no convincing relationship between lung cancer risk and frequent consumption of smoked, salted, cured or pickled foods.

Table 117: Estimates of relative risk for frequent consumption of smoked/salted/cured/pickled food

Study	Population	Food item	Relative risk (95% limits
Koo (1988)	Hong Kong women	Dried/salted fish	0.95
		Smoked/cured meat/poultry	0.92
		Pickled vegetables	1.60

References

1. Koo LC (1988) Dietary habits and lung cancer risk among Chinese females in Hong Kong who never smoked. Nutr Cancer, 11, 155-172.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

118. Smoky coal

Details of the only study which presented data relevant to an investigation of the possible association between lung cancer risk and use of smoky coal are given in Table 118. Two relative risks were estimated, 4.0 for men and 3.7 for women. In addition, evidence of an association between smoky coal use and lung cancer mortality rate per 100,000 population was presented, with rates ranging from 174.21 in an area where 100.0% of households burned smoky coal, down to 3.81 and 2.08 in two areas where no households did.

The data presented in the table provide quite strong evidence of an association between lung cancer risk and use of smoky coal, and it was suggested by the authors that in areas where smoking prevalence is low the burning of smoky coal may be the prime determinant of lung cancer [1]. However, it should be borne in mind that the data has been taken from one study only, and results from further studies are needed before definite conclusions can be reached.

Table 118: Estimates of relative risk/mortality rate for smoky coal

Study	Population	% Households burning smoky coal	Relative risk (95% limits)
He et al (1990)	Chinese men	-	4.0
	Women	-	3.7
	Chinese residents	100.0	174.21[1]
		89.7	128.31[1]
		81.9	104.09[1]
		78.0	22.96[1]
		76.1	39.46[1]
		49.7	13.48[1]
		35.2	7.49[1]
		34.0	19.03[1]
		2.7	9.55[1]
		0.0	3.81[1]
		0.0	2.08[1]

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Footnote to Table 118

1 Mortality rate per 100,000 population

References

1. He X, Chapman RS, Yang R et al (1990) Lung cancer and indoor air pollution in Xuan Wei, China: Current progress. In: Kasuga H (ed.) Indoor air quality, 435-441. Springer-Verlag.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

119. Social class

Table 119 gives details of the available data relevant to an investigation of the possible association between lung cancer risk and social class, according to the index of social class used.

For education, both of the studies using this index of social class found evidence of an increasing risk of lung cancer with decreasing educational level.

Respondents were classified according to their income in eight studies, with four finding an inverse relationship with lung cancer risk and another reporting a very weak relationship in this direction. Two studies actually observed a decrease in risk in lower income groups, although both of these studies divided the respondents into two groups only, while the remaining study failed to find any relationship between income and lung cancer risk.

None of the six studies which used occupational group as an index of social class found unequivocal evidence of an inverse relationship with lung cancer risk, although four did show that subjects in some of the "lower" occupational groups had an increased risk of lung cancer compared to those in "higher" groups.

Four studies grouped subjects according to the average rent in their area of residence, but although all found some evidence of an increasing risk of lung cancer with decreasing rent paid, in none was it a smooth relationship.

Of the 21 studies which classified respondents by their social class, as used in censuses and similar surveys, only two found a convincing inverse relationship with lung cancer risk. However, nine other studies presented some evidence that those in lower social classes have a higher risk of lung cancer than those in higher social classes.

Finally, socio-economic status was used to group respondents by seven studies, of which three found an inverse relationship with lung cancer risk, and two others found at least some evidence of such a relationship.

It can be seen from the table that a variety of indices were used to measure the respondents' social class, with some being more satisfactory than others. While occupational group and educational level are reasonably reliable, as they are based on data relating to the actual

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subject, socio-economic level, income and rent may be less so. This is because for these indices classification was often based on the average for the area of residence, rather than on that pertaining to the subject specifically. Thus, such measures could be somewhat inaccurate if the subject was not average in these respects.

Although there does appear to be some evidence that those in higher social classes are at less risk of lung cancer than those of lower social class, such a relationship is far from proved.

Table 119: Estimates of relative risk/standardized mortality ratio for low social class

Population	Standardized mortality ratios				
Education level:					
	College	High school	Elementary school	<8 years education	
US white men, 1960	61	95	114	118	
US white women, 1960	90	94	96	123	
Income level:					
	High				Low
US men, 1959-67	79	93	107	84	104[1]
US white men, 1947	67	78	99	118	134[1]
US white women, 1947	98	88	98	99	126[1]
US non-white men, 1947	-	-	45	114	127[1]
US white men, 35-54, 1959-61	23.0	25.2	36.8	48.0	60.8[2]
US white men, 55+ 1959-61.	87.0	178.7	202.0	255.0	331.7[2]
US white women, 35-54 1959-61		6.5		4.5[2]	
US white women, 55+ 1959-61		29.9		18.1[2]	

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Table 119 continued

Occupation groups:

	Professional	Managerial	Clerical	Sales	Craftsmen	Operatives	Services	Labs. exc. farm	Farmers
US men, 1950	83	94	95	103	130	107	123	127	54

	Outdoor workers		Indoor workers		
	Agriculture	Constructive	Industry	Transport	Office, etc
German men, 1958-63	58	56	176	192	135[2]

	Independent		Employees				SW	UW	Unoccupied	
	Rural	Urban	I	II	III	IV			Inc HW	Exc HW
Danish men, 1970-5	44	107	51	68	107	116	135	113	181	-
Danish women 1970-5	-	107	-	-	101	102	-	115	115	99

	Professional	Managerial	Sales	Clerical	Craftsmen	Operatives	Laborers	Services
US white men, 1972-5	83	113	91	101	111	102	113	117[1]
US white women, 1972-5	103	213	73	104	92	62	177	95[1]

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Table 119 continued

Rent level:

	High				Low
Danish men, 1943-7	66	99	90	97	116[1]
Danish women, 1943-7	50	90	79	100	131[1]
US white men, 1948-52	35.8	31.1	44.8		57.9[3]
US white women, 1948-52	5.0	6.3	5.0		7.4[3]

Social class:

	I	II	III	IV	V	Farmers	Farm workers
US men, 1949-51	85	77	112	108	112	94	54

	I	II	III	IV	V
UK men, 1911	94	106	106	83	111
UK men, 1921	100	109	97	79	124
UK men, 1931	107	96	101	91	112
UK married women[4], 1931	100	100	110	82	91
Scottish men, 1949-53	104	81	115	86	109[5]
UK men, 1951	81	82	107	91	118
UK married women[4], 1951	119	95	102	98	96
UK single women, 1951	75	101	112	100	91
Scottish men, 1959-63	61	70	104	98	151[5]
Scottish married women[4], 1959-63	59	81	78	85	138[5]
Scottish single women, 1959-63	130	73	110	174	149[5]
UK men, 1961	53	72	107	104	148
UK married women[4], 1961	83	89	102	101	131
UK single women, 1961	108	118	110	106	127
Finnish men, 1969-72	53	89	124	153	82[6]
Finnish married women[7], 1969-72	88	105	123	123	60[6]

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Table 119 continued

Norwegians, 1970-73	81	113	74	122	40
UK men, 1971	53	68	110	123	143
UK married women[4], 1971	73	82	112	125	134
UK single women, 1971	82	92	123	129	114

Socio-economic level:

	High		Low
US men, 1935-49	88	84	116[1]
US women, 1935-49	82	66	139[1]
US white men, 1949-51	35.1	43.2	47.3[8]
US white women, 1949-51	5.6	6.6	7.3[8]
Australian men, 1971	41		52[8]
Australian women, 1971	9		8[8]
Turkish men, 1979-84*	1.0	1.0(0.7-1.4)	0.9(0.7-1.3)[9]

HW = housewives; SW = skilled workers; UW = unskilled workers

* From Dosemeci et al (1993); all other studies from Logan (1982)

1 Standardized incidence ratio

2 Death rate per 100,000

3 Standardized incidence rate per 100,000

4 According to husband's occupation

5 Tumours of respiratory system

6 Comparative mortality factor

7 According to own or husband's occupation

8 Standardized death rate per 100,000

9 Relative risk

References

1. Dosemeci M, Hayes RB, Vetter R (1993) Occupational physical activity, socioeconomic status, and risks of 15 cancer sites in Turkey. Cancer Causes and Control, 4, 313-321.

2. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

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120. Soots/chimney sweeps

Although exposure to soots may occur amongst heating-unit service personnel, brick masons and helpers, building demolition personnel, insulators and firemen, chimney sweeps are the principal workers receiving occupational exposure to soots. It was thus decided to combine the reports for soots and chimney sweeps to avoid unnecessary repetition. The studies which investigated the potential risks of soot exposure and employment as a chimney sweep and lung cancer are detailed in Table 120. Thirteen standardized mortality ratios were given, lying in the range 86-313, of which 11 were raised. Additionally, two proportional registration ratios, of 128 and 134, were also given.

Little information was available from the studies on the exposures of the subjects, although one study did attempt to classify cumulative exposure to soots from the number of years spent working as a chimney sweep [1]. Soot is a complex mixture produced by the combustion of coal, coke, oils, wood, paper, rubber, plastics, and resins, and has been shown to contain potentially carcinogenic metals such as arsenic, chromium, cadmium and nickel, as well as sulphur dioxide, carbon monoxide, asbestos and organic solvents [1,2]. Additionally, the character of the sweeping duties may vary considerably and this will affect the exposure of the subject. For example, an hour's climb in chimney ducts may give an exposure to particulate dust 100 to 1000 times higher than that in other duties. Therefore, it will be very difficult to estimate the exposures of the study subjects, or the effects of any one component of soot.

Even so, IARC still felt that there was "sufficient" evidence for the carcinogenicity of soots to humans [2], although no attempt was made to identify particular causative agents from among the constituent substances in soots. It would appear that employment as a chimney sweep should also be viewed as an occupation which carries a potential carcinogenic risk.

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Table 120: Estimates of standardized mortality ratio for exposure to soot/employment as a chimney sweep

Study	Location	Subset	Standardized mortality ratio
Kennaway and Kennaway (1936, 1947)[1]	E/W	1921-1932	169($p<0.05$)[2]
		1933-1938	90($p<0.05$)[2]
Kupetz (1966)[1]	GDR		>100
OPCS (1978)	E/W	Men aged 15-64[3], 1970-2	109
		Aged 65-74, 1970-2	116
		Aged 15-74, 1966-7	134($p<0.01$)[4]
		Aged 15-74, 1968-9	128($p<0.01$)[4]
Hogstedt et al (1982)[1]	Sweden		232($p<0.05$)[2]
Logan (1982)	UK	Men, 1951	164
		1961	123
		1971	109
		Married women[5] - 1971	86
Hansen (1983)[1]	Denmark		313($p<0.05$)[2]
Hogstedt et al (1983)[1]	Sweden		277($p<0.05$)[2]
Evanoff et al (1993)	Sweden		206(154-269)

E/W = England/Wales; GDR = German Democratic Republic

1 From International Agency for Research on Cancer (1985)

2 Estimated from data given

3 Includes charwomen, office cleaners, window cleaners, chimney sweeps

4 Proportional registration ratio

5 According to husband's occupation

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

References

1. Evanoff BA, Gustavsson P and Hogstedt C (1993) Mortality and incidence of cancer in a cohort of Swedish chimney sweeps: An extended follow up study. Br J Ind Med, 50, 450-459.

2. International Agency for Research on Cancer (1985) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 35: Polynuclear aromatic compounds, part 4, bitumens, coal-tars and derived products, shale-oils and soots, 219-241. IARC, Lyon.

3. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London.

4. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

121. Spices

Only one study could be found which attempted to relate the risk of lung cancer to consumption of spices and it is detailed in Table 121. It was reported that bronchial cancer cases used less spices than controls.

With so little data, it is not possible to determine whether a protective effect of spices exists regarding lung cancer risk.

Table 121: Observations for spice consumption

Study	Population	Observations
Denoix et al (1958)	French men	Bronchial cancer cases consumed less spices than controls

References

1. Denoix PF, Schwartz D and Anguera G (1958) French investigation into the etiology of broncho-pulmonary cancer. Detailed analysis. Bull Assoc Franc pour l'etude du cancer, 49: 1-37.

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122. Sugar

Details of the only study found which investigated the possible relationship between lung cancer risk and sugar consumption are given in Table 122. It was observed that cancer cases consumed more sugar than controls.

In the absence of other supporting evidence it is not possible to evaluate the potential carcinogenicity of sugar to humans.

Table 122: Observations on sugar consumption

Study	Population	Observations
Denoix et al (1958)	French men	Bronchial cancer cases consumed more sugar than controls

References

1. Denoix PF, Schwartz D and Anguera G (1958) French investigation into the etiology of broncho-pulmonary cancer. Detailed analysis. Bull Assoc Franc pour l'etude du cancer, 49: 1-37.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

123. Sugarcane farmers

Table 123 gives details of the only study found which contained data relevant to the investigation of lung cancer risk among sugarcane farmers. A relative risk of 2.4 was estimated.

The occurrence of cases of mesothelioma among the study population led to the suggestion that exposure to fibrous cane dust may be responsible for the increased rates of lung disease observed [1], but no objective measures of the level of such dust were made by the study. Results from more studies will be needed before the carcinogenicity of exposures associated with sugarcane farming can be evaluated.

Table 123: Estimate of relative risk for sugarcane farmers

Study	Location	Relative risk (95% limits)
Rothschild and Mulvey (1982)[1]	USA	2.4

1 From Williams Pickle (1984)

References

1. Blot WJ (1984) Lung cancer and occupational exposures. In: Lung cancer: Causes and prevention, 47-64. Verlag Chemie International Inc.

2. Williams Pickle L, Correa P and Fontham E (1984) Recent case-control studies of lung cancer in the United States. In: Lung cancer: Causes and prevention, 101-115. Verlag Chemie International Inc.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

124. Taking saunas

Table 124 gives details of the only study found which attempted to relate lung cancer risk to sauna usage. A relative risk of 1.7 was found for men who were heavy smokers.

It was suggested that the increase in lung cancer risk observed may be due to the production of polycyclic aromatic hydrocarbons during the wood heating, and to asbestos, used as insulation, in the ambient air. However, in the absence of any other data, it is not really possible to evaluate the potential carcinogenicity of saunas.

Table 124: Estimate of relative risk for taking saunas

Study	Population	Relative risk
Tenkanen et al (1985)	Finnish male heavy smokers	1.7

References

1. Tenkanen L, Hakulinen T, Hakama M and Saxen E (1985) Sauna, dust and migration as risk factors in lung cancer among smoking and non-smoking males in Finland. Int J Cancer, 35, 637-642.

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

125. Talc

Table 125 details the seven studies which attempted to relate lung cancer risk to exposure to talc. Five standardized mortality ratios were calculated, and these ranged from 46-290, with all but one being raised. Two relative risks, of 1.00 and 3.24, were also estimated.

Only two studies (Kleinfeld, Selevan) provided objective data on levels of exposure to talc, although these were estimated in the study by Selevan. It is possible that inaccuracies due to misclassification of exposure could have occurred in the other studies. In addition, talc deposits contain various other minerals, including carbonates, free silica, serpentines (including chrysotile) and amphibole minerals (asbestiform and non-asbestiform), which may be potential carcinogens themselves. All of the studies appeared to have considered this to some extent, and gave information on the content of other minerals, although three studies noted that there was no exposure to asbestos (Leophonte, Rubino, Selevan). However, as most of the exposures to other minerals occurred in conjunction with exposure to talc, it was not possible to analyze the effects of talc alone. Therefore, the observed associations may have been brought about by exposure to one of these other minerals.

When evaluating the carcinogenicity of talc to humans, IARC felt that the evidence for talc not containing asbestiform fibres was "inadequate", while that for talc containing asbestiform fibres was "adequate" [2,3]. Meanwhile, a review of lung cancer and occupational exposures described talc as only a "possible" carcinogen, although no attempt was made to classify talc containing and not containing asbestiform fibres separately [1].

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Source: https://www.industrydocuments.ucsf.edu/docs/mmbl0000

Table 125: Estimates of standardized mortality ratio/relative risk for exposure to talc

Study	Population	Standardized mortality ratio
Kleinfeld et al (1974)[1]	US talc miners/millers	3.24*[2]
Rubino et al (1976)[1]	Italian talc miners/millers	46[2]
Brown et al (1979)[1]/Dement et al (1980)[1]	US talc miners/millers	290($p<0.05$)[3]
Selevan et al (1979)[1]	US talc miners/millers	163($p>0.05$)[2]
Stille and Tabershaw (1982)[1]	US talc miners/millers	157
Leophonte et al (1983)[1]	French talc workers	1.00*
Thomas and Stewart (1987)[4]	Pottery workers	143

* Relative risk

1 From International Agency for Reseach on Cancer (1987a)

2 Estimated from data given

3 Tumours of respiratory system

4 From International Agency for Reseach on Cancer (1987b)

References

1. Blot WJ (1984) Lung cancer and occupational exposures. In: Lung cancer: Causes and prevention, 47-64. Verlag Chemie International Inc.

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126. Tea drinking

Table 126 gives details of the six studies which attempted to relate the risk of lung cancer to tea consumption. Seven relative risks were estimated, ranging from 0.84-2.74, of which five were above 1.00. In addition, two studies reported finding a positive correlation between lung cancer mortality, or incidence, and tea consumption.

Over 400 volatile compounds have been identified in black teas, compared to some 200 in green teas, but with the exception of the study by Tewes et al, no attempt was made to distinguish between the different types. Furthermore, information on the consumption of other hot beverages was not given by any of the studies. Therefore, non-drinkers of tea could be drinkers of other hot beverages, such as coffee.

In the conclusions of their study, Tewes et al described tea as a "low risk agent" for lung cancer in humans [3], while IARC felt there was "inadequate evidence" for the carcinogenicity to humans of tea drinking [2].

Table 126: Estimates of relative risk for tea drinking

Study	Population	Relative risk (95% limits)
Stocks (1970)	Women in 20 countries	Positive correlation between lung cancer mortality and tea consumption
Hirayama (1974)	Japanese men	0.84
Armstrong and Doll (1975)[1]	Women in 23 countries	Positive correlation between lung cancer incidence and tea consumption
Dean et al (1977)[2]	UK men	1.22($p<0.01$)
	Women	1.65($p<0.01$)
Kinlen et al (1988)	UK men	1.4

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Table 126 continued

Tewes et al (1990)	Hong Kong women - black tea	1.43(0.88-2.33)
	Green tea	2.74(1.10-6.80)
Oguni et al (1992)[3]	Japanese residents	<1.00

1 From International Agency for Research on Cancer (1991)

2 From Tewes et al (1990)

3 From Yang and Wang (1993)

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2. International Agency for Research on Cancer (1991) Monographs on the evaluation of carcinogenic risks to humans. Volume 51: Coffee, tea, mate, methylxanthines and methylgloyoxal, 41-197. IARC, Lyon.

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127. Tetrachloroethylene

Only two studies attempted to relate the risk of lung cancer to exposure to tetrachloroethylene, and details of these are given in Table 127. Both reported an excess of lung cancers but detailed results were not given for either study.

It should be noted that laundry and dry-cleaning workers may also be exposed to other solvents, especially trichloroethylene, and petroleum solvents. However, neither study gave any information on the workers' exposures. Therefore, IARC's classification of the evidence for the carcinogenicity of tetrachloroethylene to humans as "inadequate" is not really surprising [1].

Table 127: Estimates of standardized mortality ratio for exposure to tetrachloroethylene

Study	Population	Standardized mortality ratio
Blair et al (1979)[1]	Laundry/dry-cleaning workers	>100[2]
Duh and Asal (1984)[1]	US laundry/dry-cleaning workers	>100[2]

1 From International Agency for Research on Cancer (1987)

2 No details of results given

References

1. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: an updating of IARC monographs volumes 1 to 42, 355-357. IARC, Lyon.

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128. 2,3,7,8-Tetrachlorodibenzo-para-dioxin

Table 128 gives details of the five studies which attempted to relate the risk of lung cancer to exposure to 2,3,7,8-tetrachlorodibenzo-para- dioxin (TCDD). One study estimated a relative risk of 1.41, while another calculated a standardized mortality ratio of 111. A total of five cases of lung or bronchogenic cancer were observed in the other three studies.

Two of the studies appeared to have taken objective measurements of exposure to TCDD. In the study by Manz, TCDD concentrations in samples of precursor materials, products, waste and soil from the grounds of the plant, and in the adipose tissue of volunteers were measured, while Fingerhut analyzed serum levels of TCDD. However, these measurements only appeared to provide information on current or very recent exposure to TCDD, and historical exposure data were not available from any of the studies. It is possible, then, that inaccuracies could have been introduced due to misclassification of exposure.

While IARC classified the evidence for the carcinogenicity of TCDD to humans as "inadequate" [3], Manz et al considered the results of their study and that by Fingerhut to be supportive of the hypothesis that TCDD is a human carcinogen [4].

Table 128: Estimates of relative risk/standardized mortality ratio for exposure to 2,3,7,8-tetrachlorodibenzo-para-dioxin

Study	Population	Relative risk (95% limits)
Jirasek et al (1973,1974)[1]	Czech workers	2 bronchogenic cancers observed
Thiess and Goldman (1977)[1]	German factory workers	1 lung cancer observed
Pazderova-Vejlupkova et al (1981)[2]	Chloracne patients	2 lung cancers observed
Fingerhut et al (1991)	US chemical plant workers	111(89-137)*
Manz et al (1991)	German chemical plant workers	1.41(0.95-2.01)

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Footnote to Table 128

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1977)

2 From International Agency for Research on Cancer (1987)

References

1. Fingerhut MA, Halperin WE, Marlow DA et al (1991) Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. N Engl J Med, 324, 212-218.

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3. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 350-354. IARC, Lyon.

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129. Textile manufacturing

Table 129 gives details of the numerous studies which attempted to relate exposures in the textile manufacturing industry to the risk of lung cancer.

For general textile workers, eleven relative risks were estimated, ranging between 0.3 and 4.8, with seven of them being above 1.00. Six standardized mortality ratios (SMR) were calculated, lying in the range of 87-192, with three of these being raised. Another six studies gave proportional mortality ratios, of between 43 and 130, again with three being raised. Finally, four standardized proportional incidence ratios were given, ranging from 75-91. It can be seen that these results are somewhat equivocal, with no obvious pattern of increasing or decreasing risk being apparent.

Several of the studies gave results restricted to workers in the cotton-textile industry only. From these studies five estimates of relative risk, of 0.3-0.8, were estimated along with 15 SMRs, ranging from 27.3-117, of which only two were raised. These results are consistent with a decrease in the risk of lung cancer among respondents whose work exposes them to cotton-textiles only.

A wide variety of occupational health hazards are present in the textile industry, of which the most common is organic dust, particularly cotton dust. Other exposures include sizing agents and spinning oils used during spinning and weaving, bacteria, desiccants and defoliants found in raw cotton, pesticide residues found in raw wool, and a variety of bleaching, scouring, singeing and mercerizing agents used during fabric preparation. Chemicals are also widely used during dyeing, printing and finishing, and exposures may be highly complex [3]. It was suggested by one of the studies [7] where an increased mortality was observed that it may have been due to exposure to asbestos and mineral oils, but as little information was available from any of the studies about the nature of the subjects' exposures, it is difficult to ascertain the level of exposure, or to estimate the effects of any one substance. Also, as an increase in the risk of lung cancer was not generally seen in the evidence for textile workers it is possible that the results of the study by Zappa may have been due to chance alone.

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A number of hypotheses have been put forward to try to explain the apparent decrease in mortality in workers in the cotton-textile industry. Firstly, it has been suggested that cotton-textile workers have a lower rate of smoking than the general population, due in part to the fact that for many years such employees were not permitted to smoke at work-because of an explosion hazard associated with vegetable dusts [2]. However, it has been reported that the level of smoking among cotton-textile workers is comparable to that of respondents in other occupations [2,6]. The second possible explanation is that exposure to cotton dust may stimulate stimulate mucus production, which might protect against carcinogens such as cigarette smoke [2,3,6]. A third hypothesis is based on evidence that endotoxins, whilst toxic in high concentrations, may be potent anticancer agents at the levels at which they are found in airborne cotton dust [2,3,6].

IARC [3] classify as "limited" the evidence that exposures in the textile industry as a whole increase the risk of cancer, and indeed the pattern of results reported is unimpressive. However, the possibility of a protective effect for workers in cotton-textiles appears to be much better supported.

Table 129: Estimates of relative risk/standardized mortality ratio for textile workers

Study	Population	Relative risk (95% limits)
General textile workers:		
Versluys (1949)[1]	Dutch male weavers/woollens workers	43[2]
Guralnik (1963)[1]	US male textile mill products labourers	192*
Ashley (1967)[1]	UK wool producing boroughs	87*
Menck and Henderson (1976)[1]	US male textile workers	119(p>0.05)*
Williams et al (1977)[1]	US male textile mill products workers	2.6

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Table 129 continued

Decoufle et al (1978)[1]	US male textile mill operatives	1.5(p>0.05)
Harrington et al (1978)[1]	US male textile workers	0.88
Newhouse (1978)[1]	UK male dyers/bleachers/textile workers	94*[3]
Buiatti et al (1979)[1]	Italian textile workers - male	2.0(1.4-2.7)
	Females	4.8(0.9-14.0)
Delzell and Grufferman (1983)[1]	US female textile workers	90[2]
Milne et al (1983)[1]	US male textile manufacturers	1.9
Coggon et al (1986)[1]	UK male textile workers	0.3
OPCS (1986)	British textile workers - male	94*
	Married women[4]	115*
Siemiatycki et al (1986)[1]	Wool workers	0.5(0.3-1.0)
	Synthetics workers	0.5(0.2-1.1)
Olsen and Jensen (1987)[1]	Danish male textile workers	88(70-111)[5]
	Females	82(56-120)[5]
	Male spinners/weavers/finishers	91(66-125)[5]
	Females	75(39-143)[5]
Paci et al (1987)[1]	Italian male nonasbestos textile workers	1.5(0.98-2.3)
Dubrow and Gute (1988)[1]	US male textile workers	<100[2]
	Service workers	130(p>0.05)[2]
	Synthetics/silk only workers	130(p>0.05)[2]
O'Brien and Decoufle (1988)[1]	US male carpet/textile workers	100(90-110)[2]
Zappa et al (1993)	Italian residents	1.45(1.0-2.1)
Cotton-textile workers:		
Kennaway and Kennaway (1936)[1]	E/W male cotton spinners/piecers	0.3
	Cotton strippers/grinders/card-room jobbers	0.3
	Cotton weavers	0.5
Enterline (1965)[6]	Male cotton-textile workers	27.3*[7]
Ashley (1967)[1]	UK cotton-textile producing towns	89*
Registrar General (1971)[8]	UK cotton-textile workers - men	85(p<0.05)*
	Single women	78*
	Married women[4]	81(p<0.05)*

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Table 129 continued

Henderson and Enterline (1973)[6]	Male cotton-textile workers	54.8*[7]
Daum et al (1975)	US male cotton-textile workers	93*
	Females	96*[3]
Merchant and Ortmeyer (1981)[6]	Male cotton-textile workers	40*[7]
Logan (1982)	UK male cotton spinners - 1931	117*
	1951	57*
	1961	66*
	1971	109*
	Married women[4] - 1961	91*
Siemiatycki et al (1986)[1]	Canadian cotton workers	0.8(0.4-1.3)
Levin et al (1987)[1]	Chinese cotton-textile workers	0.7(0.6-0.9)
Hodgson and Jones (1990)[8]	British cotton workers	76(54-102)*

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1990)

2 Proportional mortality ratio

3 Estimated from data given

4 According to husband's occupation

5 Standardized proportional incidence ratio

6 From Enterline et al (1985)

7 Tumours of respiratory system

8 From Rylander (1990)

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130. Tin miners

Three studies were found which attempted to relate lung cancer risk to employment as a tin miner, and details of these are given in Table 130. Two relative risks, of 1.01 and 2.4, were estimated and one standardized mortality ratio of 157.7 was calculated.

It was suggested that underground tin miners may be exposed to various substances, including arsenic, silica, radon and polycyclic aromatic hydrocarbons (PAHs) [1-3]. The study by Hodgson and Jones used contemporary measurements of radon to estimate historical levels, and also recorded exposure to arsenic among the subjects. McLaughlin et al measured silica concentrations as well as gathering limited information on exposure to radon, arsenic, PAHs, asbestos, nickel, talc and cadmium. The study by Wu et al measured radon levels, and dust concentrations of arsenic, iron, chromium, cadmium and nickel. However, as little information was available on lung cancer risk in relation to such exposures, and with so few studies reporting, the exact carcinogen(s) responsible for the observed increases in lung cancer risk remain unclear.

Table 130: Estimates of relative risk/standardized mortality ratio for tin miners

Study	Population	Relative risk (95% limits)
Wu et al (1989)	Chinese tin miners	2.4
Hodgson and Jones (1990)	English tin miners	157.7($p<0.01$)*
McLaughlin et al (1992)	Chinese tin miners	1.01(0.77-1.33)[1]

* Standardized mortality ratio

1 Estimated from data given

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3. Wu K-G, Fu H, Mo C-Z, and Y L-Z (1989) Smelting, underground mining, smoking, and lung cancer: A case-control study in a tin mine area. Biomed Environ Sci, 2, 98-105.

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